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# Pseudotumor cerebri presenting with 2<sup>nd</sup> and 7<sup>th</sup> nerve palsy

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# **ABSTRACT**

Idiopathic intracranial hypertension, alternatively called pseudotumor cerebri is characterized by increased intracranial pressure of unknown pathology with the absence of any structural lesions. This intracranial hypertension most commonly presents with postural headache, nausea, vomiting, transient visual loss with cranial nerve involvement, with most common VI cranial nerve involvement. In the following case report, we present a pseudotumor cerebri presenting with 2<sup>nd</sup> and 7<sup>th</sup> nerve palsy.

Keywords: Hypertension, Nerve Palsy, Pseudotumor Cerebri

# 1. INTRODUCTION

Idiopathic intracranial hypertension, alternatively called pseudotumor cerebri is a disorder of unknown pathology, which proves any absence of intracranial space occupying lesions or CSF obstruction (Biousse et al., 2012; Albalawi et al., 2021). This IIH is most commonly seen among women of childbearing age mainly in overweight and obese women. It is also been associated with drugs like tetracycline and with OCPs and even with high dose vitamin A derivatives. Patients who are affected with IIH 92% present with headache, 72% with visual obsurations, 59% with multiple cranial nerve involvement (Peralta & Cestari, 2018). The most commonly involved cranial nerve among them is 6th CN in 12% adults and 40% in children. Very less frequently other nerves are being involved. Very verylimited number of cases has been reported with 2<sup>nd</sup> and 7<sup>th</sup> nerve palsy (Acheson, 2006) and even there are even fewer cases reported with 2nd nerve involvement. In the light of this, we present a very rare case with raised ICT involving 2nd nerve and unilateral 7th nerve involvement. This will alert clinicians to suspect IIH in patients with these cranial nerve involvements.

# 2. CASE REPORT

A 32 year old female came to the ER with the complaints of complaints of headache and giddiness for past 1 week, complaints of blurring of vision for the past 1 week which was gradual in onset, complaints of inability to close right eyes, complaints of diplopia, complaints of deviation of angle of mouth to the left, patient had no complaints of slurring of speech, no complaints of weakness of both upper and lower limbs, no complaints of seizures, vomiting,



trauma. She did not have any long-standing history of headaches, fever or any history of oral contraceptive pills or other significant drug history.

On examination patient is conscious oriented, afebrile, moderately built and nourished with BMI 24. No pallor, icterus, cyanosis, clubbing, pedal edema, lymphadenopathy. Her Vitals: HR: 110/min, BP: 140/90 RR:18/min, Spo2:99% in RA. Systemic examination: CNS- Higher mental function normal, 2<sup>nd</sup> cranial nerve shows B/L distant 6/18, B/L color vision lost, 3<sup>rd</sup>, 4<sup>th</sup>, 6<sup>th</sup> cranial nerve extra ocular movement full, pupil size b/l 3mm equal, relative afferent pupil defect (RAPD), 7<sup>th</sup> cranial nerve showed a deviation of angle of mouth to the right side, inability to close left eye, drooling of saliva present, loss of nasolabial fold in left side present all other cranial nerve examinations were absolutely normal, her motor and sensory system were normal, no cerebellar signs, no signs of nystagmus. Other system examination: RS; BAE+ no added sounds, CVS: S1S2+ P/A; soft, no organomegaly.

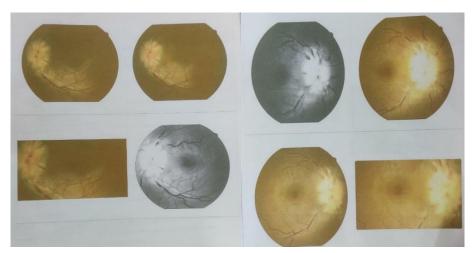


Figure 1 B/L fundus showing established papilloedema

Fundus examination showed bilateral disc swelling with cirumpapillary hemorrhages (Figure 1). The above findings were confirmed with OCT (Optical Coherence Tomography) of the optic disc. There was severe diffuse thickening of the nerve fibre layer. The thickness of NFL in right and left eye was 240mcm and 220mcm. Investigations showed CBC (TC:7560, HB: 12, PLT: 2.6 lakh/cumm), ESR:40, CRP:0.6. Her LFT and RFT were within normal limits. Urine routine was normal. Above all, her CT and MRI did not show any significant abnormality.

At day 1 of her admission guarded lumbar puncture was done which showed a opening CSF pressure was more than 35cmH20 and her cerebral fluid analysis showed (protein: 27mg/dl, glucose: 71mg/dl). Therefore a diagnosis of idiopathic intracranial hypertension was made with involvement of 2<sup>nd</sup> and 7<sup>th</sup> nerve palsy.

We started the patient oh oral steroids (methypredisolone 40mg BD), we started her on T.acetozolamide 250mg qid and also furosemide was added to her management. Even after putting her on all these drugs and even with guarded lumbar puncture did not improve her symptoms and even her papiloedema, therefore we planned for a the coperitoneal shunt and not for a ventriculo peritoneal shunt because her ventricles were so much dilatated in imaging. After the coperitoneal shunt her symptoms subsided with mild residual papilloedema, her visual field defects were normal and patient was discharged.

# 3. DISCUSSION

We present a case of moderately built 32 year old female with complaints of headache, giddines, blurring of vision for past 1 week. Here CNS examination showed involvement of second and seventh nerve palsy and her fundus examination showed established papilloedema (Wakerley et al., 2015). Other conditions like lymes disease, sarcoidosis should be ruled out before making a diagnosis of raised ICT because in these conditions also the most common sole nerve to be affected in raised ICT is 7th nerve. All imaging MRI/CT brain should to taken to come to a clear diagnosis of raised ICT (Wall & George, 1991). IIH affects predominantly moderately built to obese individuals with incidence of 11.9 per 100,000 per year.

The pathophysiology of 2nd and 7th nerve palsy in IIH is not yet clearly known and there is no satisfactory hypotheses exist. The extra pressure is known to act on the extra axial facial nerve (Soroken et al., 2016). This theory is very well accepted in seventh and sixth nerve palsy, due to the known fact that sixth nerve having a long course and the most common nerve affected in ICT. But the cause of second nerve palsy getting affected is still a hypothesis. The only supporting hypothesis is that elevation of the

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intracranial pressure causes compression of the optic nerve which impairs its axoplasmic flow and causing 2nd nerve palsy and papilloedema (Rangwala & Liu, 2007). There are only very few cases reported with isolated facial nerve palsy and only one or two cases reported with 2nd nerve palsy.

# 4. CONCLUSION

Idiopathic intracranial hypertension should to always suspect in young obese women, who comes to our OPD with headache and transient loss of vision. The diagnosis of IIH is always being a diagnosis of conclusion other obstructive or structural lesions should be ruled out. Here by our case we would like to notify that raised ICT can also occur with unilateral 7<sup>th</sup> nerve palsy and very rare presentation with 2<sup>nd</sup> nerve palsy. The mechanism of this raised ICH is still not clear with 7<sup>th</sup> and 2<sup>nd</sup> nerve palsy. And still prompt investigations and various studies are needed to prove its actual hypothesis.

#### Informed consent

Consent has been obtained from the patient with regards to the use of the pictures for publication.

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#### Conflicts of interest

The authors declare that there are no conflicts of interests.

## Data and materials availability

All data associated with this study are present in the paper.

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